APPENDIX E

RISK ANALYSIS

Valley Paving & Asphalt

Facility ID No. 777-00086

P-060024

Health Risk Assessment for Valley Paving and Asphalt, Inc.

January 25, 2007

State of Idaho
Department of Environmental Quality
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Attachment to the Valley Paving and Asphalt, Inc. Permit to Construct Application for Modification to their Portable Hot Mix Asphalt Plant Located in McCall, Idaho
Permit Number P-060024

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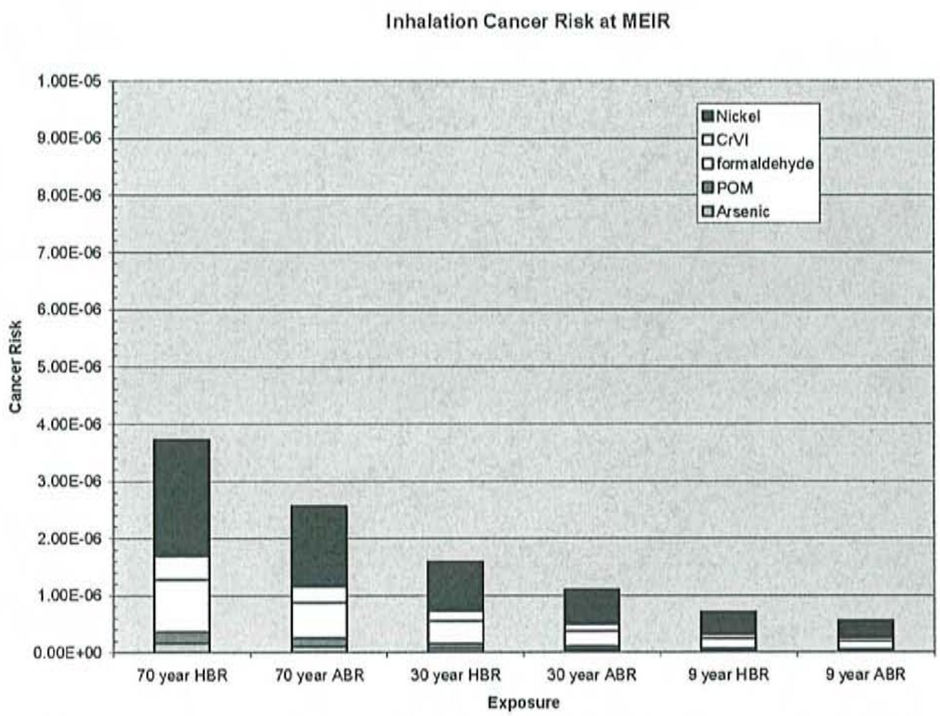

Figure C.1. Inhalation risk at MEIR location for contaminants of concern. (HBR = high breathing rate, and ABR = average breathing rate.)

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Abbreviations, Acronyms, and Symbols

AACC	acceptable ambient concentrations for carcinogens
ABR	average breathing rate
AE	average exposure
Cal/EPA	California Environmental Protection Agency
cm^2	square centimeters
DEQ	Idaho Department of Environmental Quality
EPA	U.S. Environmental Protection Agency
HBR	high breathing rate
HE	high exposure
HSPRAG	Hot Spots Program Risk Assessment Guidelines
IDAPA	Idaho Administrative Procedures Act
IUR	inhalation unit risk
kg	kilogram
m^3	cubic meters
MEIR	maximum exposed individual resident
MEIW	maximum exposed individual worker
μg	micrograms
NATA	National Air Toxics Assessment
PMI	point of maximum impact
POM	polycyclic organic matter
TAP	toxic air pollutant

Executive Summary

Poor air quality is a concern for both citizens and environmental and public health agencies. It can have an impact on the health of individuals and may adversely impact sensitive populations such as children, the elderly, and those with asthma, respiratory disease, and heart disease. McCall, Idaho is not exempt from air quality problems. Periods of poor air quality in McCall may be due in part to population growth, an increase in vehicle use, factory emissions, wood stove burning, and forest fires.

A risk assessment is a scientific process that estimates the type and level of human health risk posed by exposure to chemicals. This health risk assessment was undertaken to characterize the risks associated with the emissions from the asphalt plant of Valley Paving and Asphalt, Inc. in McCall. Recent public interest in the permit granted by the Department of Environmental Quality (DEQ) to the Valley Paving hot mix asphalt plant prompted DEQ to look at the overall risk associated with emissions from the facility.

Computer modeling of the plant's air emissions indicates that carcinogens (cancer-causing toxic air pollutants) may have the most potential to pose a health risk. This health risk assessment, then, focuses on those particular carcinogens and attempts to determine their impacts on human health. This study does not address the odor emissions from Valley Paving. While odor may have a significant impact on personal well-being and quality of life, there is no objective way to characterize odors and their impact.

Key Findings

Key findings of this health risk assessment are described below.

Pollutants of Concern

Computer modeling of Valley Paving's air emissions determined that five pollutants exceeded or were close to exceeding state regulatory exposure limits. These five pollutants are:

- polycyclic organic matter (POM)
- formaldehyde
- nickel
- chromium 6+
- arsenic

These pollutants were analyzed further in order to characterize potential risk to public health.

Conclusions

The findings of this assessment, which focus on the potential residence and work locations predicted to be the most impacted by emissions, are that the levels of toxic air pollutants from Valley Paving are considered to be low and are not considered to pose an unreasonable risk to human health. Exposure from breathing to all but one of the contaminants (when considered individually) is not considered to increase the chance of developing cancer. The one exception is nickel, which shows a very small increased cancer risk for those who breathe contaminated air for 24 hours per day for 70 years at a high breathing rate. (Note a high breathing rate is assumed to be more reflective of active lifestyles. In this risk assessment both a high breathing rate and an average breathing rate are considered.) However, since the risk is very low, DEQ does not consider it to be a public health hazard.

In order to adequately describe the full impacts of the emissions from Valley Paving, methods of exposure (other than through inhalation) to the five pollutants were also considered. For example, some of the air pollutants could be deposited onto the ground, allowing possible human exposure through soil contact. However, when the additional exposure pathways are considered and the risks from each pollutant are added together, the total risk is considered low and DEQ does not consider it to be a public health hazard.

If you have questions or comments, please contact the Department of Environmental Quality at 208-373-0502.

Introduction

This section gives background information on the Valley Paving facility, its pollutants, and basic concepts of health risk assessments.

Background

Poor air quality is a concern for both citizens and environmental and public health agencies. It can have an impact on the health of individuals and may adversely impact sensitive populations such as children, the elderly, and those with asthma, respiratory disease, and heart disease. McCall, Idaho is not exempt from air quality problems. Periods of poor air quality in McCall may be due in part to population growth, an increase in vehicle use, factory emissions, wood stove burning, and forest fires.

This health risk assessment was undertaken to characterize the risks associated with the emissions from the asphalt plant of Valley Paving and Asphalt, Inc in McCall. Recent public interest in the permit granted by the Department of Environmental Quality (DEQ) to the Valley Paving hot mix asphalt plant in McCall prompted DEQ to look at the overall risk associated with emissions from the facility.

Computer modeling of the plant's air emissions indicated that carcinogens (cancer-causing toxic air pollutants) have the most potential to pose a health risk. This health risk assessment focuses on those particular carcinogens and attempts to determine their impacts on human health. This assessment, however, does not address the odor emissions from Valley Paving. While odor may have a significant impact on personal well-being and quality of life, there is no objective way to characterize odors and their impact.

Facility Information

Table 1 gives basic information about the facility.

Table 1. Valley Paving and Asphalt facility information.

Facility Name	Valley Paving and Asphalt, Inc.
Address	Portable facility located approximately 1 mile west of Highway 55 along Deinhard Lane
ID Number	777-0086
Operations	See Section 2, "Facility Description," of the Statement of Basis for Valley Paving and Asphalt Permit to Construct (attached to this document)
Emitted Substances	See Appendix B, "Emissions Inventory," of the Statement of Basis for Valley Paving and Asphalt (attached to this document)

Pollutants of Concern

Computer modeling of Valley Paving's air emissions (see the Modeling Memo in Appendix C of the Statement of Basis for the Valley Paving and Asphalt Permit to Construct, attached to this document) indicated that possible emissions of four pollutants exceeded the screening level acceptable ambient concentrations for carcinogens (AACC) listed under DEQ toxics air pollution rules (DEQ 2007). The four pollutants are:

- polycyclic organic matter (POM)
- formaldehyde
- nickel
- chromium 6+

The modeled concentrations of the pollutants analyzed are shown in Table 2.

Table 2. Results of the facility-wide toxic air pollutant modeling analysis for Valley Paving.

Toxic Air Pollutant (TAP)	Averaging Period	Maximum Modeled Concentration (μg/m3)[1]	Acceptable Ambient Concentrations for Carcinogens (AACC) (μg/m3)	% of AACC
Polycyclic organic matter (POM)	Annual	0.00052	0.00030	173
Arsenic	Annual	0.00015	0.00023	64
Nickel	Annual	0.019	0.0042	283
Chromium 6+	Annual	0.00011	0.000083	130
Formaldehyde	Annual	0.12	0.077	160

[1] Micrograms per cubic meter

To better quantify the cancer risk associated with the emissions of Valley Paving, a more comprehensive health risk assessment, as described in this document, was performed. This health risk assessment evaluated additive effects of the four carcinogens listed above. Arsenic, whose concentration was close to the AACC limit (64% of the AACC), was also analyzed as part of the assessment. The assessment also, where appropriate, evaluated additional pathways for exposure (dermal and oral), in addition to the inhalation pathway.

Cancer Health Impacts and Multi-Pathway Substances

Cancer health impacts are normally expressed in terms of a risk value. Concentrations of contaminants are compared to concentration levels that are associated with specific risks, which are considered *reference* levels. A screening risk value of a 1 in a million risk level is commonly used by regulatory authorities. If exposure is less than the screening value, then adverse health effects are unlikely.

An important factor to consider for carcinogens is the time period of exposure. If exposure to a chemical occurs for 70 years (considered to represent an average lifetime exposure) at a 1 in a million risk level, this means that one would expect to observe one excess cancer occurrence (over background) for a population of one million people. In addition to the 70-year exposure level, 9-year and 30-year exposure levels are also considered. The 9-year and 30-year levels represent less than lifetime exposures, applicable for individuals who do not spend their entire lives in the area in question.

For carcinogens, the U.S. Environmental Protection Agency (EPA) has a target protection level at the 1 in a million risk but also assumes that 100 in a million (or 1 in 10,000) is also an acceptable exposure level(EPA 2006). In the DEQ Risk Evaluation manual, a 10 in a million (or 1 in 100,000) total additive risk level for all of the pollutants of concern is considered acceptable (DEQ 2005).

For certain pollutants, exposure can occur through pathways other than inhalation. For example, if a pollutant is deposited onto soil, it could be ingested with soil by children playing in the dirt or by residents eating unwashed, locally-grown produce. Such a pollutant is considered a multi-pathway substance. Four of the five pollutants in this assessment are considered multi-pathway substances: POM, arsenic, nickel, and chromium 6+. However, although nickel and chromium 6+ are subject to deposition, they have not been found to be carcinogenic through oral exposure, and therefore, only the cancer risk from inhalation is calculated for these two pollutants (Cal/EPA 2003).

Health Risk Assessment Procedures

The results of the exposure modeling conducted for this health risk assessment predict the concentrations of chemicals of concern emitted from the Valley Paving facility in McCall. A description of various sources of air emissions from the facility, along with the results of exposure modeling (including assumptions, model and meteorological data, etc.) conducted for this health risk assessment are provided in the Modeling Memo in Appendix C of the Statement of Basis for the Valley Paving and Asphalt Permit to Construct (attached to this document).

Guidelines Used

The guidelines used for this health risk assessment are those used by the California Environmental Protection Agency (Cal/EPA) in their Air Toxics Hot Spots Program Risk Assessment Guidelines (HSPRAG). Facilities in California are required to regularly perform this type of risk assessment, and the procedures have gone through extensive peer review.

Cal/EPA's HSPRAG is taken from *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments.* The guidance manual, which can be downloaded from www.oehha.ca.gov, is a summary of four extensive technical support documents for California's Hot Spots Program. In summary, the manual is "a concise description of the algorithms, recommended exposure variates, and cancer and non-cancer health values needed to perform a health risk assessment" (Cal/EPA 2003, p. ES-1).

One departure this health risk assessment took from HSPRAG was to use EPA's risk guidelines, when available, instead of those determined by Cal/EPA. The reasons for this departure are: (1) when the EPA inhalation unit risk values are identical to those contained in the Idaho rules for these substances, and (2) when multiple sets of oral cancer risk values exist (as they did in this case), the values established by EPA are, on a nationwide basis, normally given primacy over the others (EPA 2003).

Pathways Considered

The paragraphs below describe the various pathways that toxic air pollutants can take to enter the body: (1) the inhalation pathway, and (2) various other pathways, which are grouped under the category of multi-pathway.

Inhalation Pathway Substances

Following HSPRAG, three compounds (nickel, chromium 6+, and formaldehyde) from Valley Paving's air emissions were analyzed for cancer risk based on an inhalation pathway only. Formaldehyde is in this group because its impact is only through inhalation and is not considered a multi-pathway substance; in other words, it is not subject to deposition based on its physical properties.

Although chromium 6+ and nickel could be viewed as multi-pathway contaminants, there is no definitive data that indicates they are carcinogenic via an oral pathway (Cal/EPA 2003). Following HSPRAG, this health risk assessment based the cancer risk associated with these two compounds on an inhalation pathway only. However, HSPRAG also recommends that chromium 6+ and nickel be considered for non-carcinogenic oral health effects (in other words, exposure via an ingestion pathway). See Appendix A for these results.

Multi-Pathway Substances

The remaining two compounds from Valley Paving's air emissions (POM and arsenic) were analyzed for cancer risk via inhalation and exposure through other pathways (dermal, soil ingestion, and ingestion of home-grown produce). Other possible pathways that were not evaluated were exposure from fish ingestion; water ingestion; meat, milk, and egg ingestion; and breast milk ingestion. Fish ingestion and water ingestion pathways were eliminated due to the lack of standing water near the Valley Paving facility. It was also assumed that livestock and animal farming for food does not exist next to the Valley Paving facility, eliminating meat, milk, and egg ingestion. For chemicals to be considered for the breast milk ingestion pathway, they need to be bio-accumulative (in other words, they must have the capability of becoming concentrated in human adipose tissue and breast milk lipid). HSPRAG recommends evaluating the breast milk pathway for PCBs, dioxins, and furans, which are all well documented to be bioaccumulative (Cal/EPA 2003), but not for POM or arsenic.

Subpopulations

A possible subpopulation of concern in the area of Valley Paving is children. Children may have more exposure to contaminants due to their activities (for example, playing in soil) and higher breathing and ingestion rates for their body size compared to adults. For carcinogenic effects which are based on lifetime exposures (the 70-year exposure level), lifetime risk factors are designed to be protective of both adults and children. To address short-term exposure of children to possible contaminants, 9-year exposure levels include factors that are more representative of children and their behavior.

Dose-Response Assessment

Dose-response assessment describes the probability of adverse health effects (known as the response) when a person or persons are exposed to certain concentrations (known as the dose). Individual dose-responses for POM, arsenic, nickel, chromium 6+, and formaldehyde will be discussed later in this section, along with the specific possible cancers associated with each compound (referred to as carcinogenic endpoints).

Calculating Cancer Risk

For carcinogens, the health guidance value used to determine a substance's toxicity is the cancer potency factor (also known as the slope factor). To determine cancer risk, the cancer potency of a substance is multiplied by the average dose (usually determined from a modeling analysis), as shown in Equation 1.

Equation 1:

$$\text{Cancer Risk} = (\textit{inhalation dose}\,({}^{mg}\!/_{kg-day})) \times (\textit{Cancer Potency}\,({}^{mg}\!/_{kg-day})^{-1})$$

The dose units of mg/kg-day represent milligrams of contaminant per kilograms of bodyweight per day. To convert potential cancer risk to chances per million of developing cancer, one multiplies the potential risk by 10^6 or 1 million.

The default analysis in this health risk assessment was performed assuming a worst-case scenario of a 70-year (or lifetime) exposure level with a high-end exposure rate (for example, breathing rate). Assuming a 70-year exposure level is considered very conservative. (Superfund assessments are normally performed assuming a 30-year exposure.) Additional exposure levels (30-year and 9-year) and exposure rates (average) were also considered in this health risk assessment.

Calculating Cancer Potency

For inhalation, the risk factor often referenced is the inhalation unit risk (IUR) (when the slope factor is based on a concentration instead of a dose). To convert the IUR to an inhalation cancer potency factor, the equation below can be used:

Equation 2:

$$\text{Cancer potency} = {}^{IUR \times 70kg \times CV}\!/_{20m^3}$$

where

IUR = inhalation unit risk (in $\mu g/m^3$)
70 kg = reference human body weight (154 pounds)
CV = conversion factor from mg to μg (1000)
20 m^3 = reference breathing rate/day

POM Dose-Response

POM is a broad class of organic compounds formed as by-products of incomplete combustion. POM is also referred to as PAH (polycyclic aromatic hydrocarbons), which is a subset of POM. One of the most well-characterized compounds of the POM (and PAH) class is benzo(a)pyrene. It is often used as a surrogate for other PAH compounds in modeling analyses. As discussed in Appendix G of HSPRAG, some PAHs are more "potent" in their cancer-causing ability and others are less or much less "potent" (Cal/EPA 2003, G-1). According to Cal/EPA, based on available carcinogenic data for PAHs, the use of benzo(a)pyrene as a surrogate for PAHs would probably overestimate cancer risk and therefore be more protective of public health (Cal/EPA 2003).

Lung cancer has been shown to arise from human exposure to POM, though the carcinogenity of any specific POM chemical is impossible to determine from human studies. However, risk factors associated with benzo(a)pyrene have been determined from animal studies, which indicate that benzo(a)pyrene is a carcinogen by both oral and inhalation routes. The oral cancer endpoints from animal studies indicate that exposure to benzo(a)pyrene can lead to stomach, larynx, or esophageal cancer.

For benzo(a)pyrene, EPA has determined an oral slope factor of 7.3 mg/kg-day (EPA 2007), used to calculate cancer risk from oral ingestion. To calculate cancer risk from inhalation of benzo(a)pyrene, EPA does not provide an IUR value; therefore, both the Cal/EPA IUR value of 0.0011 $\mu g/m^3$ and the IDEQ value of 0.0033 $\mu g/m^3$ were considered. The more protective IDEQ value was used in this health risk assessment to determine an inhalation slope factor of 3.85 $(mg/kg\text{-}day)^{-1}$.

Arsenic Dose-Response

Based on data from human studies (mainly occupational studies), arsenic is considered a known human carcinogen. Studies of workers in copper smelters found increased mortality resulting from lung cancer due to inhalation exposure to arsenic. From studies of populations with high levels of arsenic in drinking water, there is also evidence that oral intake can lead to cancers of internal organs (kidney, liver, lung, and bladder), as well as skin cancer.

For arsenic, EPA has determined an oral slope factor of 1.5 mg/kg-day, used to calculate cancer risk from oral ingestion. To calculate cancer risk from inhalation of arsenic, EPA has determined an IUR value of 0.0043 $\mu g/m^3$ (EPA 2007), which corresponds to an inhalation slope factor of 15.1 $(mg/kg\text{-}day)^{-1}$.

Nickel Dose-Response

Nickel is a known human carcinogen from inhalation exposure. The two observed endpoints are lung and nasal cancer. Most occupational human studies were performed on workers at nickel refineries where the nickel that was inhaled was a combination of nickel compounds (for example, nickel subsulfide and nickel oxide). This combination of nickel compounds is called nickel refinery dust. Results from animal results also indicate that some nickel refinery dusts may be carcinogenic. It is difficult to determine the exact speciation of nickel from industrial processes (such as, asphalt production), but the assumption that the nickel exposure is similar to that of nickel refinery dust should be the most conservative and protective of human health. There is insufficient data to determine whether nickel is a carcinogen from oral exposure. Therefore, this health risk assessment (as described in the HSPRAG guidelines) only considers exposure to nickel via inhalation (Cal/EPA 2003).

To calculate cancer risk from inhalation of nickel, EPA has determined an IUR of 0.00024 $\mu g/m^3$ (EPA 2007). This corresponds to an inhalation slope factor of 0.84 $(mg/kg\text{-}day)^{-1}$.

Chromium 6+ Dose-Response

Chromium 6+ (or hexavalent chromium) is a known human carcinogen from inhalation exposure. Consistent results have been obtained from occupational studies that correlate chromium exposure to lung cancer. Chromium exists naturally in two different forms, chromium 3+ and chromium 6+. Since animal studies indicate only chromium 6+ is a carcinogen, chromium 6+ is assumed to be the only form of chromium that is a human carcinogen. There is insufficient data to determine whether chromium 6+ is a carcinogen from oral exposure.

Therefore, this health risk assessment (as described in the Cal/EPA guidelines) only considers exposure to chromium 6+ via inhalation (Cal/EPA 2003).

To calculate cancer risk from inhalation of chromium 6+, EPA has determined an IUR value of 0.012 $\mu g/m^3$ (EPA 2007). This number corresponds to an inhalation slope factor of 42 $(mg/kg\text{-}day)^{-1}$.

Formaldehyde Dose-Response

Based on limited data in humans and sufficient data from animals, formaldehyde is considered a probable human carcinogen. Limited human data come from occupational or residential exposure to formaldehyde where an increased mortality from lung or nasopharyngeal cancer was observed. There are more comprehensive data from animal studies that correlate inhalation exposure to occurrence of cancer. There is insufficient data to determine whether formaldehyde is a carcinogen from oral exposure. Therefore, this health risk assessment (following HSPRAG guidelines) only considers exposure to formaldehyde via inhalation (Cal/EPA 2003).

To calculate cancer risk from inhalation of formaldehyde, EPA has determined an IUR value of 0.000013 $\mu g/m^3$ (EPA 2007). This number corresponds to an inhalation slope factor of 0.0455 $(mg/kg\text{-}day)^{-1}$.

Inhalation Risks

As described in Equation 1, the dose and the cancer potency both need to be determined to calculate risk. The IUR values from EPA (or Cal/EPA for POM) and Idaho DEQ are listed in Table 3. Using Equation 2, the inhalation cancer potency factor was also calculated and is listed in the table below.

Table 3. Summary of IUR values used in this health risk assessment.

Toxic air pollutant (TAP)	IUR (EPA)[1]	IUR (Cal/EPA)[2]	IUR (DEQ)[3]	Cancer Potency Factor (inhalation)	Cancer Potency Factor (ingestion) (EPA)[4]
POM		0.0011	0.0033	3.85	7.3
Arsenic	0.0043		0.0043	15.1	1.5
Nickel	0.00024		0.00024	0.840	None
Chromium 6+	0.012		0.012	42.0	
Formaldehyde	0.000013		0.000013	0.0455	

[1] www.epa.gov/iris (EPA 2007)
[2] HSPRAG (Cal/EPA 2003)
[3] IDAPA 58.01.01.586 (DEQ 2007)
[4] www.epa.gov/iris (EPA 2007)

The Idaho IUR values are identical to the EPA IUR values (except for POM). Both the IUR values for POM from DEQ and Cal/EPA were considered; however, since the IUR from DEQ is more protective of human health, it was used in this assessment.

Carcinogenic Endpoints

As discussed in the preceding dose-response sections, each contaminant of concern is associated with specific possible cancers, known as carcinogenic endpoints. These endpoints are listed in Table 4.

Table 4. Carcinogenic endpoints for contaminants of concern.

Toxic Air Pollutant (TAP)	Inhalation Carcinogenic Endpoints	Oral Carcinogenic Endpoints
POM	Lung cancer	Stomach, larynx, esophageal cancer
Arsenic	Lung cancer	Kidney, liver, lung, bladder, skin cancer
Nickel	Lung, nasal cancer	N/A
Chromium 6+	Lung cancer	N/A
Formaldehyde	Lung, nasopharyngeal cancer	N/A

Exposure Locations Considered

To adequately address the risk associated with the emissions from Valley Paving, locations representing three types of exposure were analyzed:

- PMI (the point of maximum impact, or the highest off-site location of risk)
- MEIR (maximum exposed individual resident, or the residential location off-site that has the highest risk)
- MEIW (maximum exposed individual worker, or the workplace off-site that has the highest risk)

This health risk assessment focused on the MEIR and MEIW locations, since they have human receptors.

Locations representing all three types were determined as described in the Modeling Memo in Appendix C of the Statement of Basis for the Valley Paving and Asphalt Permit to Construct (attached to this document). These points are indicated in the map shown in Figure 1. Due to the uncertainty in the modeling work, a buffer of 200 meters (indicated by the yellow circle around each point) was used.

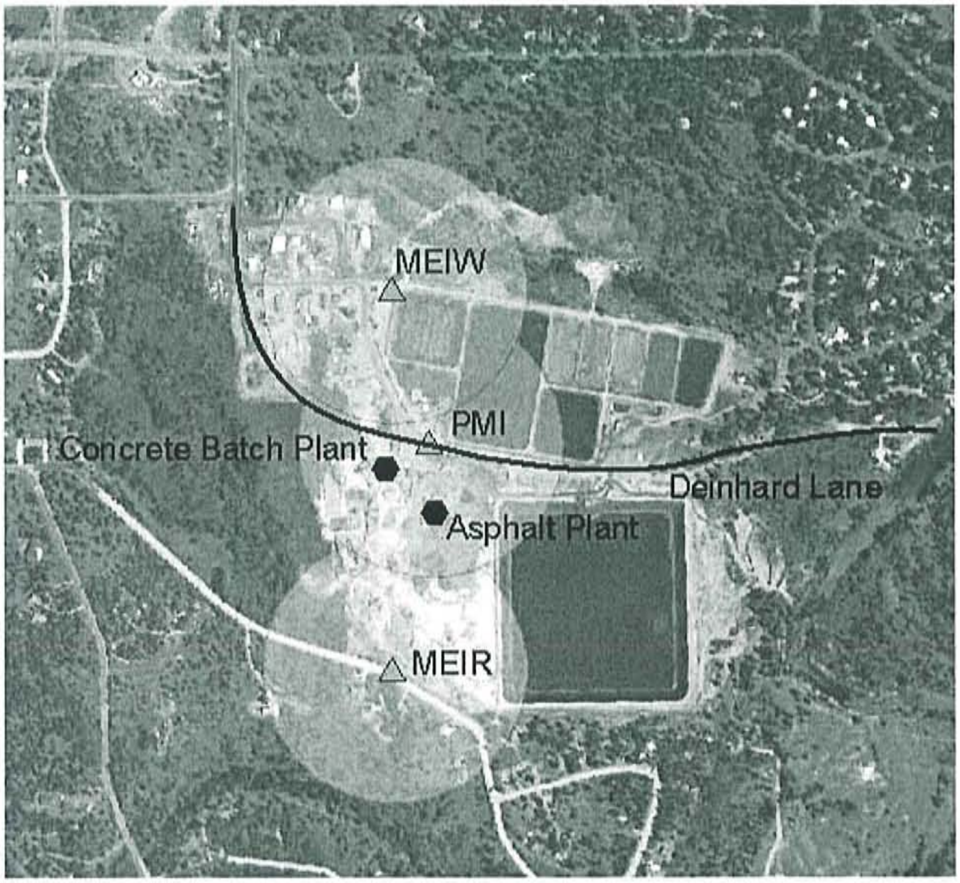

Figure 1. Locations of PMI, MEIW, and MEIR.

The map in Figure 1 was based on a 2004 National Agriculture Imagery Program photo and does not contain the road Deinhard Lane, along which the PMI is located. Deinhard Lane was hand-drawn on the map in black.

For the PMI, MEIR, and MEIW locations, the total 70-year high-end risk is shown in Table 5 below.

Table 5. Maximum 70-year total risk for locations analyzed.

Risk	Point of Maximum Impact (PMI)[1]	Maximum Exposed Individual Resident (MEIR)	Maximum Exposed Individual Worker (MEIW)
70-Year High-End Cancer Risk	0.0000093	0.0000062	0.0000023

[1] Inhalation risk only

At the locations of human exposure (MEIR and MEIW), the total estimated cancer risk is less than 10 in a million. For the PMI location, only the risk due to inhalation was calculated. It is less than the 10 in a million risk level, but it is unrealistic to consider this site as a possible off-site 70-year human exposure location since no one would be living there. A more detailed description of the risks associated with the PMI location is included in Appendix B.

Modeled cancer risk results for the MEIR and MEIW locations are described in greater detail below. In this health risk assessment, POM and arsenic were treated as multi-pathway pollutants for the MEIR and MEIW locations.

Maximum Exposed Individual Resident (MEIR) Modeling Results

The MEIR location is directly south of the Valley Paving hot mix asphalt facility, along Chad Drive (Figure 1). To determine this location, the highest inhalation risks associated with residential areas were analyzed. Modeled concentrations of the pollutants of concern at this location are shown below in Table 6.

Table 6. Air toxics concentrations at the MEIR location.

Toxic Air Pollutant (TAP)	Concentration ($\mu g/m^3$)[1] at MEIR Location
POM	0.000052
Arsenic	0.0000029
Nickel	0.0064
Chromium 6+	0.000026
Formaldehyde	0.053

[1] Micrograms per cubic meter

Both inhalation risk and risk associated with additional pathways were evaluated for the MEIR location. The inhalation risk results for this location are shown in Table 7 below.

Table 7. Inhalation cancer risk associated with the chemicals of concern at the MEIR location.

Inhalation Exposure Levels	POM	Arsenic	Nickel	Chromium 6+	Formaldehyde	Sum of Cancer Risk due to Inhalation
70-year HBR[1]	0.000000203	0.000000163	0.00000203	0.000000404	0.000000912	0.000003715
70-year ABR[2]	0.000000140	0.000000113	0.00000140	0.000000278	0.000000629	0.000002562
30-year HBR	0.000000087	0.000000070	0.00000087	0.000000173	0.000000391	0.000001592
30-year ABR	0.000000060	0.000000048	0.00000060	0.000000119	0.000000270	0.000001098
9-year HBR	0.000000039	0.000000031	0.00000039	0.000000077	0.000000173	0.000000706
9-year ABR	0.000000030	0.000000024	0.00000030	0.000000060	0.000000135	0.000000549

[1] HBR = high breathing rate
[2] ABR = average breathing rate

The largest contributor to inhalation risk at the MEIR location is from nickel. It is the only compound for which the risk level exceeds 1 in a million. The sum of inhalation cancer risk values for all the pollutants of concern at the MEIR location is less than 5 in a million. Total risk values associated with a sum of inhalation and oral/dermal risk (associated with multi-pathway contaminants) are shown below in Table 8.

Table 8. Total cancer risk associated with the chemicals of concern at the MEIR location.

Total Risk Multi-Pathway	
Inhalation Exposure Levels	*Sum of Cancer Risk due to All Pathways*
70-year HE[1]	0.0000062
70-year AE[2]	0.0000032
30-year HE	0.0000027
30-year AE	0.0000014
9-year HE	0.0000012
9-year AE	0.0000007

[1] HE = high exposure
[2] AE = average exposure

The total cancer risk from all five toxic air pollutants emitted at the MEIR location is less than 10 in a million.

Maximum Exposed Individual Worker (MEIW) Modeling Results

The MEIW location is north of the Valley Paving hot mix asphalt facility, close to Industrial Loop (Figure 1). To determine this location, the closest off-site work place sites to Valley Paving were located. Modeled concentrations of the pollutants of concern at this location are shown below in Table 9.

Table 9. Air toxics concentrations at the MEIW location.

Toxic Air Pollutant (TAP)	Concentration ($\mu g/m^3$)[1] at MEIR Location
POM	0.000068
Arsenic	0.000088
Nickel	0.0053
Chromium 6+	0.000066
Formaldehyde	0.044

[1] Micrograms per cubic meter

Both inhalation risk and risk associated with additional exposure pathways (dermal, soil ingestion, and home-grown produce ingestion) were evaluated for the MEIW location. The inhalation risk results are shown in Table 10 below. For the MEIW location, Cal/EPA makes no differentiation between high and average exposure levels (Cal/EPA 2003).

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Table 10. Inhalation cancer risk associated with the chemicals of concern at the MEIW location.

Inhalation Exposure Levels	POM	Arsenic	Nickel	Chromium 6+	Formaldehyde	Sum of Cancer Risk due to Inhalation
70-year	0.00000011	0.00000013	0.00000044	0.00000028	0.00000020	0.00000116
30-year	0.00000005	0.00000006	0.00000019	0.00000012	0.00000009	0.00000050
9-year	0.00000001	0.00000002	0.00000023	0.00000004	0.00000003	0.00000032

The largest contributor to inhalation risk at the MEIW location is from chromium 6+. All calculated inhalation risk values are less than 5 in a million, and for no single chemical does the risk level exceed 1 in a million at the MEIW location. Total risk values associated with a sum of inhalation and oral/dermal risk (associated with multi-pathway contaminants) are shown below in Table 11.

Table 11. Total cancer risk associated with the chemicals of concern at the MEIW location.

Total Risk Multi-Pathway	
Inhalation Exposure Levels	*Sum of Cancer Risk due to All Pathways*
70-year	0.0000023
30-year	0.00000097
9-year	0.00000046

All calculated risk values for the five toxic air pollutants emitted at the MEIW location are less than 5 in a million.

Risk Characterization

Risk was characterized by determining contaminant concentrations for the pollutants of concern emitted from the Valley Paving facility, calculating doses, and calculating possible exposures at selected locations.

Contaminant Concentrations

To characterize the health risk associated with the Valley Paving facility, equations contained in HSPRAG (Cal/EPA 2003) were used to calculate risk levels based on the modeled concentrations of contaminants. Specifically, the equations outlined in Section 5 of HSPRAG were entered into an Excel spreadsheet to make calculations easier to perform. A detailed description of the calculations for each exposure pathway is given below.

Air Concentrations

Air concentrations were obtained directly from the dispersion modeling results as described in the Modeling Memo in Appendix C of the Statement of Basis for the Valley Paving and Asphalt Permit to Construct (attached to this document). This was the only value needed to calculate cancer risk for nickel, chromium 6+, and formaldehyde.

Soil Concentrations

For the multi-pathway substances (POM and arsenic), concentrations in other media like soil also needed to be calculated. The soil concentration was assumed to be a function of deposition, length of time for accumulation, half-life of chemical of interest, mixing depth, and soil density. Since the hot mix asphalt facility does have a wet venturi scrubber for particulate matter control, the deposition rate for POM and arsenic was assumed to be 0.02 meters/second (Cal/EPA 2003). The soil half-lives were 100,000,000 and 570 years for arsenic and POM (as benzo(a)pyrene), respectively. All other values were default values recommended by HSPRAG (Cal/EPA 2003). Two soil concentrations were calculated: one for a playground setting (used for soil ingestion and dermal pathways) and the other for an agricultural setting (used for the vegetable intake pathway). The difference between the two soil concentrations is the soil mixing depth (in other words, the depth of soil disturbance by human activities). In a playground setting, the soil is disturbed to a much shallower depth (by children playing) than in an agricultural setting, where cultivation activities disturb the soil to a greater depth. The soil mixing depth for an agricultural setting is 0.15 meters versus 0.01 meters for the playground setting.

The calculated soil concentrations for arsenic were 0.095 mg/kg for a playground setting and 0.006 mg/kg for an agricultural setting. While extensive sampling of soils for arsenic has not been performed in Idaho, the United States Geological Survey's average concentration of arsenic in western soils is 5 mg/kg (Shacklette and Boerngen). Thus, the calculated soil concentration values in this health risk assessment are likely much less than the likely background (naturally occurring) arsenic concentration.

The soil concentrations for POM were calculated to be 0.0056 mg/kg for a playground setting and 0.00037 mg/kg for an agricultural setting. Definitive concentrations of POM in soil are also not available for Idaho, but soil samples in remote areas of Wyoming contained total PAH concentrations of up to 0.210 mg/kg (Agency for Toxic Substances and Disease Registry 1995). As with arsenic, the calculated values of POM in this study are significantly less than the likely background.

Water, Animal, and Fish Concentrations

Since there do not appear to be any significant standing bodies of water near the Valley Paving facility that would be used as drinking water, the exposure to the multi-pathway contaminants of concern (POM and arsenic) via water ingestion was not evaluated. This reasoning was also used to eliminate the fish ingestion pathway.

The human exposure route from eating meat, milk, and eggs was also not evaluated, on the assumption that few farm animals exist in the area immediately adjacent to the facility.

Home-Grown Produce Concentrations

The exposure route through eating vegetation was evaluated on the assumption that backyard gardens could exist in the area around the facility. The concentration of a chemical in or on vegetation is a function of direct deposition onto the vegetation and uptake from soil. For the plant exposure pathway, four types of produce were considered: exposed vegetation, leafy vegetation, protected vegetation, and root vegetation. Leafy vegetation consists of broad-leafed vegetables like spinach. Root vegetation consists of underground vegetables, which are not subject to direct deposition. Exposed vegetation is composed of vegetation with only a small area subject to direct deposition (for example, strawberries), and protected vegetation consists of produce whose edible part is completely protected from deposition (for example, nuts). According to HSPRAG, the protected and root vegetable routes do not need to be considered for POM (Cal/EPA 2003). The default parameters from HSPRAG were used for calculations of plant concentrations. The calculated concentration values for POM and arsenic in produce are shown below in Table 12.

Table 12. Plant concentration values[1] for POM and arsenic.

Type of Vegetable	POM	Arsenic
Leafy	0.092805126	0.123841075
Root	0 [2]	0.002543107
Protected	0 [2]	0.005721991
Exposed	0.046918049	0.055473611

[1] In micrograms per kilogram.
[2] Root and protected exposure not evaluated per HSPRAG guidance for POM (Cal/EPA 2003).

Breast Milk Concentration

Exposure of contaminants via mother's milk is also not appropriate for the multi-pathway contaminants being evaluated (POM and arsenic). For chemicals to be considered for the breast milk ingestion pathway, they need to become concentrated in human adipose tissue and breast milk lipid. HSPRAG only recommends evaluating the breast milk ingestion pathway for PCBs, dioxins, and furans (Cal/EPA 2003), not for POM or arsenic.

Doses

This section discusses doses of the contaminants of concern via inhalation, dermal, and ingestion exposure pathways.

Dose Calculation

Once the air, soil, and plant concentrations are determined, human exposure estimates can be calculated. The dose estimate procedure is outlined below, and results are given for 9-year, 30-year, and 70-year exposure levels for the MEIR and MEIW locations.

Inhalation Dose

For the inhalation route, exposure is a function of pollutant concentration and respiration (breathing rate). The respiration values provided in HSPRAG (Cal/EPA 2003) are included in Table 13. Note that the 9-year breathing rates are higher than the 30-year and 70-year rates, to take into account the higher breathing rates (faster respiration) of children. The breathing rates for 30 years and 70 years are assumed to reflect lifetime exposures and therefore the combination of childhood and adult exposures.

Table 13. Breathing rate estimates for 9-year, 30-year, and 70-year exposure levels and for workers.

9-Year Exposure Level		30-Year and 70-Year Exposure Levels		Off-Site Worker
Average Daily Breathing Rate[1]	*High-End Daily Breathing Rate*[1]	*Average Daily Breathing Rate*[1]	*High-End Daily Breathing Rate*[1]	*Daily Breathing Rate (Single Value)*[1]
452	581	271	393	149

[1] Daily breathing rates are in (Liters/(kilogram of body weight * day)).

The inhalation dose values for each of the contaminants of concern at the MEIR location are shown in Appendix C.

Dermal Dose

Exposure through dermal absorption is a function of the soil/dust loading onto the skin, the amount of skin exposed, and the concentration of the contaminant in the soil. The recommended point estimates for dermal exposure for soil loading, exposure frequency, and surface area exposed are provided in Table 14.

Table 14. Recommended dermal pathway values for 9-year, 30-year, and 70-year exposure levels and for workers.

	9-Year Exposure Level		30-Year and 70-Year Exposure Levels		Off-Site Worker
Body Weight (kg)	18		63		70
	Average	*High-End*	*Average*	*High-End*	*Single Value*
Soil Loading (mg/cm^2-day)[1]	0.2	1.0	0.2	1.0	1.0
Exposure Frequency (day/year)	228	350	121	350	245
Surface Area Exposed (cm^2)	2778	3044	4700	5500	5800

[1] milligrams per square centimeter per day.

The dermal dose values for each of the contaminants of concern at the MEIR location are shown in Appendix C.

Ingestion Dose

Exposure through ingestion is a function of the concentration of the contaminant in soil or vegetation (for the purposes of this health risk assessment), the gastrointestinal absorption of the contaminant, and the amount ingested. The default values from HSPRAG were used in the ingestion exposure calculations. For the plant pathway, the fraction of homegrown produce (in relation to the total amount of food eaten per person) was assumed to be 0.15 for a non-urban setting. (The fraction for an urban setting is 0.052.) The soil ingestion rates and estimates for food consumption rates (both high-end and average) were taken from HSPRAG (Cal/EPA 2003).

The ingestion dose values for each of the contaminants of concern at the MEIR location are shown in Appendix C.

Exposure Locations

This section discusses the modeled risks determined for the MEIR and MEIW locations.

Maximum Exposed Individual Resident (MEIR) Results

A bar graph illustrating the predicted cancer risks associated with the emissions from Valley Paving at the MEIR location is shown below in Figure 2. This graph shows the risks of POM and arsenic (both of which are treated as multi-pathway pollutants and contribute additional non-inhalation cancer risk), nickel, chromium 6+, and formaldehyde. As seen in the graph, the 70-year high-end exposure level has the highest cancer risk (0.0000062). The largest contributor to risk is via the inhalation route. (See Figure C.1 in Appendix C for a bar chart illustrating inhalation risk only at the MEIR location.) The second highest contributor to risk is plant ingestion. In all, the total cancer risk from all pathways is less than 10 in a million. These levels are considered to be very low, and, based on EPA air toxics risk guidelines (EPA 2006) and DEQ hazardous waste guidelines (DEQ, 2005), they do not pose a significant excess cancer risk.

Total Multi-Pathway Cancer Risk at MEIR

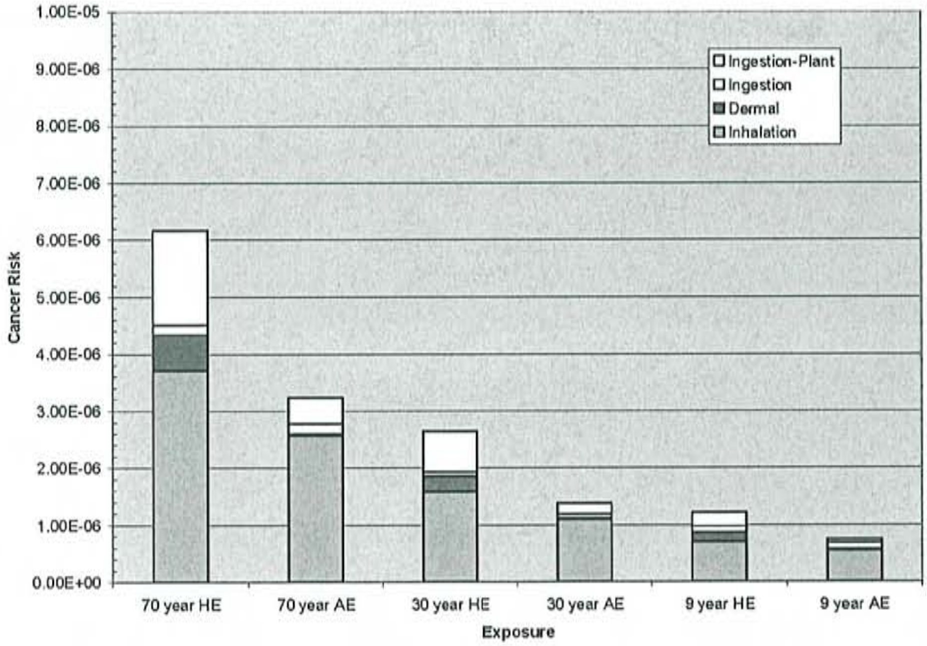

Figure 2. Sum of inhalation cancer risk and cancer risk from other pathways at the MEIR location for contaminants of concern.

(HE = high exposure, and AE = average exposure.)

Maximum Exposed Individual Worker (MEIW) Results

A bar graph showing the predicted cancer risks associated with the emissions from Valley Paving at the MEIW location is shown in Figure 3. This graph shows the risks of POM and arsenic (both of which are treated as multi-pathway pollutants and contribute additional non-inhalation cancer risk), nickel, chromium 6+, and formaldehyde. As with the MEIR location, the largest risk at the MEIW location is associated with inhalation, though dermal exposure is also a potentially significant route. In all, the total cancer risk from all pathways at the MEIW location is less than 10 in a million. These levels are considered to be very low, and, based on EPA air toxics guidelines (EPA 2006) and DEQ hazardous waste guidelines (DEQ, 2005) they do not pose a significant excess cancer risk.

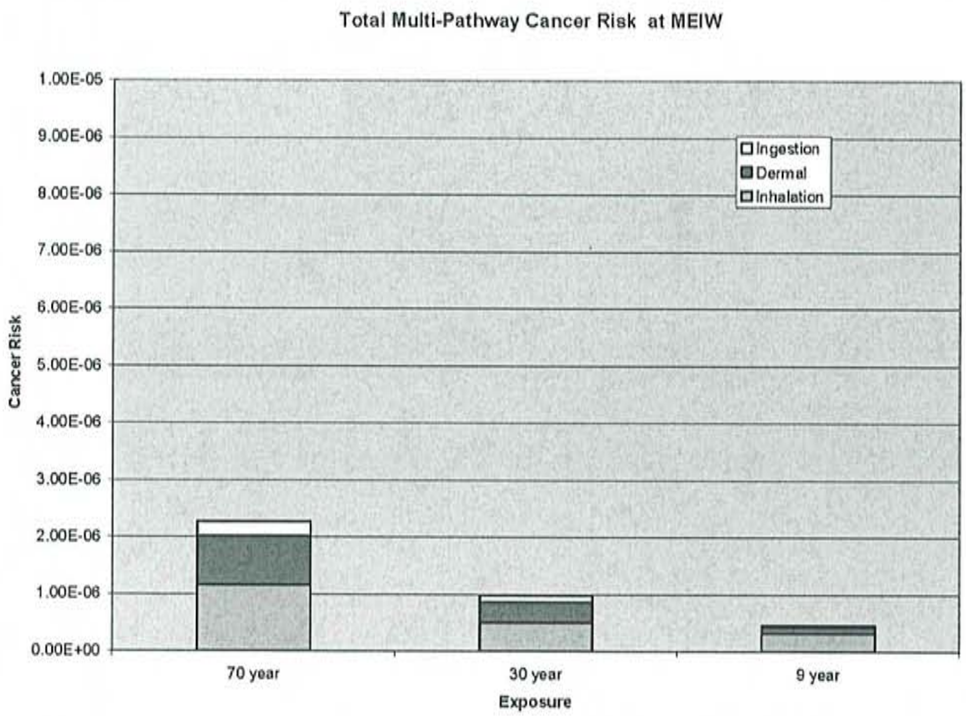

Figure 3. Sum of inhalation cancer risk and cancer risk from other pathways at the MEIW location for contaminants of concern.

Discussion

Based on the health risk assessment described in this document, the Valley Paving off-site air emissions pose a cancer risk that is greater than 1 in a million, but below 10 in a million. The risk level posed by the facility's emissions is considered low and should not pose a significant risk to human health. Inhalation exposure to all but one of the contaminants (when considered individually) is not considered to increase the chance of developing cancer. The one exception is nickel, which shows a very small increased cancer risk for those who breathe the contaminated air for 24 hours per day for 70 years at a high breathing rate. However, since the risk is very low, DEQ does not consider it to be a public health hazard.

To place the cancer risk values from Valley Paving in context, they could be compared to inhalation risk values determined for other Idaho locations. Unfortunately, Idaho DEQ has not previously conducted a comprehensive risk assessment for a facility's air emissions, nor is there any air toxics monitoring data for McCall.

While not a valid comparison to modeled worst case impacts from a single source (Valley Paving), data from the two air toxics monitoring studies that have been conducted in Idaho will be summarized for reference. The data were collected at specific locations that were assumed to be representative of a wider area. The monitoring data reflected background emissions and contributions from nearby industrial and transportation sources.

One air toxics monitoring event occurred in the Treasure Valley. In 2003, DEQ measured air toxics concentrations at a location in Nampa, Idaho and the Idaho Department of Health and Welfare analyzed the data. (Idaho Department of Health and Welfare 2007) Six compounds exceeded the one in a million reference cancer risk level in that study: formaldehyde, acetaldehyde, crotonaldehyde, arsenic, chromium, and cadmium. Four of the compounds were determined to have risk levels of ten in a million or greater. These cancer risks were not considered a public health concern. (Idaho Department of Health and Welfare 2007)

A second air toxics monitoring study occurred as part of a Superfund assessment in the Portneuf Valley. DEQ measured concentrations of metal contaminants in air from 2003-2005 in Pocatello. Non metal air toxics (or organic contaminants like formaldehyde) were not measured at the site. The concentration of one of the metals (arsenic) was slightly greater than the one in a million risk level. The Department of Health and Welfare determined the risk to be very low and the concentrations to not be a public health hazard. (Idaho Department of Health and Welfare, August 2007)

When a health risk assessment is performed, it normally includes assumptions that are more protective of public health (in other words, more conservative) in order to offset uncertainty in risk values. One conservative assumption made in this health risk assessment was that dermal exposure (for high-end exposure rates) occurs 350 days per year (see Table 14). In McCall, which is snow-covered for most of the winter, this is unrealistic. However, since dermal exposure was not a significant route of exposure in this situation, no adjustment was made for this fact. Another conservative assumption made in this health risk assessment, when analyzing the plant exposure pathway, was that no adjustments were made for the shortened growing season in McCall due to cold weather.

Even though the health risks posed by this facility are low, there are ways that concerned residents can minimize their exposure locally. Two suggestions for minimizing inhalation exposure are 1) minimize exercise next to the facility, 2) keep doors and windows closed. Since eating home-grown produce is the exposure pathway with the second greatest risk, efforts could be made to limit exposure through that pathway as well. The calculations for plant concentration indicate that most of the contaminant concentration is from direct deposition and not from uptake of contaminants into the plant. (For leafy plants, the deposition contribution is approximately 70 times greater than root uptake). A way to minimize the exposure from direct deposition would be to thoroughly wash vegetables grown in the area to remove any possible deposition of air contaminants.

If you have questions or comments, please contact the Department of Environmental Quality at 208-373-0502.

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Appendix A. Evaluation of Non-Carcinogenic Chronic Exposure to Chromium 6+ and Nickel

Although chromium 6+ and nickel could be viewed as multi-pathway contaminants, there is no definitive data that indicates that they are carcinogenic via an oral pathway. Following HSPRAG (Cal/EPA 2003), this health risk assessment based the cancer risk associated with these two compounds on an inhalation pathway only.

HSPRAG also recommends that chromium 6+ and nickel be considered for non-carcinogenic oral health effects (in other words, exposure via an ingestion pathway). To evaluate these health-effects, the hazard quotient is calculated as shown in Equation 3.

Equation 3:
Hazard quotient (oral) = exposure Pathway Dose / Chronic Oral Reference Exposure Level

The chronic oral reference exposure levels for chromium 6+ and nickel are 0.02 and 0.05, respectively.

The hazard quotient total is the sum of the individual hazard quotients for each exposure pathway, as shown below:

Hazard quotient (nickel) = HQ (Dermal) + HQ (Soil ingestion) + HQ (vegetable)

Hazard quotient (nickel) = 0.0073

Hazard quotient (chromium 6+) = 0.00011

Hazard quotients with values less than one are considered protective of human health for chronic exposures to contaminants. Since the hazard quotients calculated here are much less than one, there should be no non-carcinogenic health effects associated with exposure to either nickel or chromium 6+ from the Valley Paving facility.

Appendix B. Risks at Point of Maximum Impact (PMI) Location

Based on the modeling results, the point of maximum impact (PMI), which is the location of highest off-site risk, is along West Deinhard Lane, just outside the hot mix asphalt facility. This location is marked on the map in Figure 1. Since there is some uncertainty in the exact location of the PMI, a buffer of 200 meters around this location is also included to more accurately represent the possible concentrations. Since this area is clearly not an inhabited area, only the inhalation risk was evaluated. Along with the 70-year high-end risk, additional risk levels were also calculated. These are shown in Table B.1 below.

Table B.1. Inhalation cancer risk associated with the chemicals of concern at the PMI location.

Inhalation Exposure Levels	POM	Arsenic	Nickel	Chromium 6+	Formaldehyde	Sum of Cancer Risk due to Inhalation
70-year HBR[1]	0.00000225	0.00000041	0.00000358	0.00000096	0.00000209	0.00000929
70-year ABR[2]	0.00000155	0.00000028	0.00000247	0.00000066	0.00000144	0.00000640
30-year HBR	0.00000097	0.00000018	0.00000153	0.00000041	0.00000089	0.00000398
30-year ABR	0.00000067	0.00000012	0.00000106	0.00000028	0.00000062	0.00000274
9-year HBR	0.00000043	0.00000008	0.00000068	0.00000018	0.00000040	0.00000177
9-year ABR	0.00000033	0.00000006	0.00000053	0.00000014	0.00000031	0.00000137

[1] HBR = high breathing rate
[2] ABR = average breathing rate

The largest contribution to risk is from nickel; however, all calculated risks are less than 10 in a million.

The modeled concentrations of the chemicals of concerns at the PMI location are listed in Table B.2 below.

Table B.2. Air toxics concentrations at PMI (location of maximum risk).

Toxic Air Pollutant (TAP)	Concentrations ($\mu g/m^3$) at PMI
POM	0.00052
Arsenic	0.00015
Nickel	0.012
Chromium 6+	0.00011
Formaldehyde	0.12

The modeled concentrations of the chemicals of concerns at the PMI location are shown below in the bar graph in Figure B.1. Nickel is the largest contributor to risk at this location. While the inhalation cancer risk is greater than the 1 in a million screening level for all exposure levels, the total inhalation cancer risk is less than 10 in a million. These levels are considered to be very low, and based on EPA (EPA 2006) and DEQ hazardous waste guidelines (DEQ, 2005), they do not pose a significant excess cancer risk.

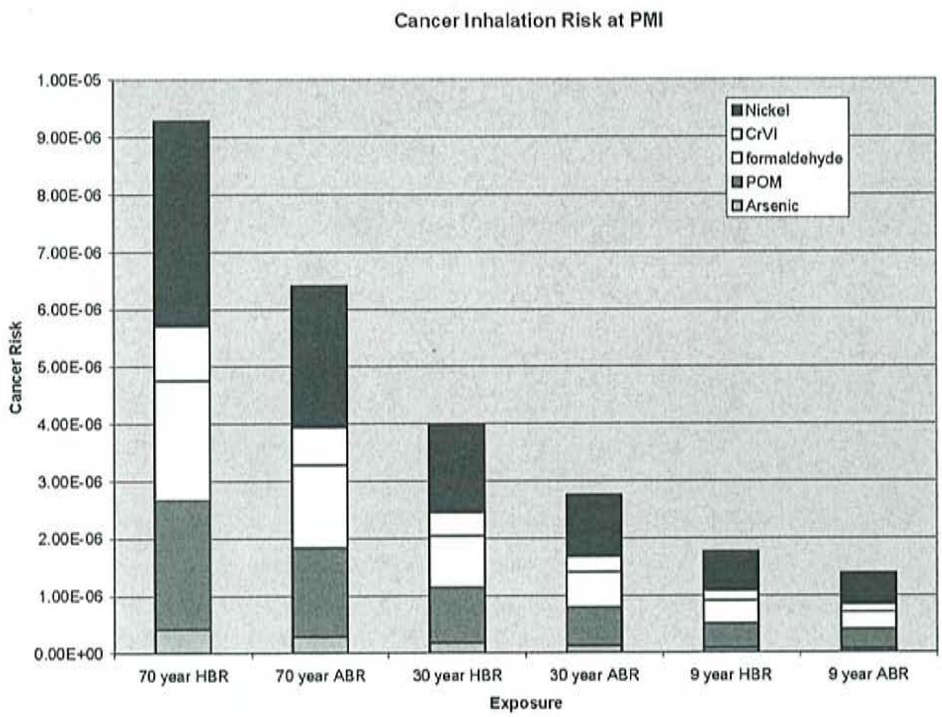

Figure B.1. Inhalation risk at the PMI location for contaminants of concern. (HBR = high breathing rate, and ABR = average breathing rate.)

Appendix C. Risks at Maximum Exposed Individual Resident (MEIR) Location

Inhalation Doses for Maximum Exposed Individual Resident (MEIR)

Tables C.1 through C.5 give the inhalation doses for POM, arsenic, nickel, chromium 6+, and formaldehyde at the MEIR location. Notes explaining the column heading abbreviations are included after Table C.5.

Table C.1. POM Inhalation Dose for MEIR

Dose-inh[1]	Cair[2]	DBR[3]	A[4]	EF[5]	ED[6]	AT[7]	Exposure
1.76E-08	4.67E-05	393	1	350	70	25550	70 HBR[8]
1.21E-08	4.67E-05	271	1	350	70	25550	70 ABR[9]
7.54E-09	4.67E-05	393	1	350	30	25550	30 HBR
5.20E-09	4.67E-05	271	1	350	30	25550	30 ABR
3.34E-09	4.67E-05	581	1	350	9	25550	9 HBR
2.60E-09	4.67E-05	452	1	350	9	25550	9 ABR

Table C.2. Arsenic Inhalation Dose for MEIR

Dose-inh[1]	Cair[2]	DBR[3]	A[4]	EF[5]	ED[6]	AT[7]	Exposure
1.09E-08	2.88E-05	393	1	350	70	25550	70 HBR[8]
7.48E-09	2.88E-05	271	1	350	70	25550	70 ABR[9]
4.65E-09	2.88E-05	393	1	350	30	25550	30 HBR
3.21E-09	2.88E-05	271	1	350	30	25550	30 ABR
2.06E-09	2.88E-05	581	1	350	9	25550	9 HBR
1.60E-09	2.88E-05	452	1	350	9	25550	9 ABR

Table C.3. Nickel Inhalation Dose for MEIR

Dose-inh[1]	Cair[2]	DBR[3]	A[4]	EF[5]	ED[6]	AT[7]	Exposure
2.42E-06	6.42E-03	393	1	350	70	25550	70 HBR[8]
1.67E-06	6.42E-03	271	1	350	70	25550	70 ABR[9]
1.04E-06	6.42E-03	393	1	350	30	25550	30 HBR
7.15E-07	6.42E-03	271	1	350	30	25550	30 ABR
4.60E-07	6.42E-03	581	1	350	9	25550	9 HBR
3.58E-07	6.42E-03	452	1	350	9	25550	9 ABR

Table C.4. Chromium 6+ Inhalation Dose for MEIR

Dose-inh[1]	Cair[2]	DBR[3]	A[4]	EF[5]	ED[6]	AT[7]	Exposure
9.61E-09	2.55E-05	393	1	350	70	25550	70 HBR[8]
6.63E-09	2.55E-05	271	1	350	70	25550	70 ABR[9]
4.12E-09	2.55E-05	393	1	350	30	25550	30 HBR
2.84E-09	2.55E-05	271	1	350	30	25550	30 ABR
1.83E-09	2.55E-05	581	1	350	9	25550	9 HBR
1.42E-09	2.55E-05	452	1	350	9	25550	9 ABR

Table C.5. Formaldehyde Inhalation Dose for MEIR

Dose-inh[1]	Cair[2]	DBR[3]	A[4]	EF[5]	ED[6]	AT[7]	Exposure
2.00E-05	5.32E-02	393	1	350	70	25550	70 HBR[8]
1.38E-05	5.32E-02	271	1	350	70	25550	70 ABR[9]
8.59E-06	5.32E-02	393	1	350	30	25550	30 HBR
5.92E-06	5.32E-02	271	1	350	30	25550	30 ABR
3.81E-06	5.32E-02	581	1	350	9	25550	9 HBR
2.96E-06	5.32E-02	452	1	350	9	25550	9 ABR

Notes for Tables C.1 – C.5:

[1] Dose-inh = inhalation dose (mg/kg/day) in scientific notation (i.e. 1.0E-02 = 0.01)
[2] Cair = air concentration (μg/m3) in scientific notation (i.e. 1.0E-02 = 0.01)
[3] DBR = daily breathing rate (Liters/kg body weight – day)
[4] A = inhalation absorption factor
[5] EF = exposure frequency (days/year)
[6] ED = exposure duration (years)
[7] AT = averaging time (e.g., 25550 days for 70 years for cancer risk)
[8] ABR = average breathing rate
[9] HBR = high breathing rate

Dermal Doses for Maximum Exposed Individual Resident (MEIR)

Tables C.6 and C.7 give the dermal doses for POM, arsenic, nickel, chromium 6+, and formaldehyde at the MEIR location. Notes explaining the column heading abbreviations are included after Table C.7.

Table C.6. Arsenic Dermal Dose for MEIR

Dose-dermal[1]	Cs[2]	SA[3]	SL[4]	Ef[5]	ABS[6]	ED[7]	BW[8]	AT[9]	Exposure
1.60E-07	47.69	5500	1	350	0.04	70	63	25550	70-year HE[10]
9.44E-09	47.69	4700	0.2	121	0.04	70	63	25550	70-year AE[11]
6.84E-08	47.69	5500	1	350	0.04	30	63	25550	30-year HE
4.04E-09	47.69	4700	0.2	121	0.04	30	63	25550	30-year AE
3.98E-08	47.69	3044	1	350	0.04	9	18	25550	9-year HE
4.73E-09	47.69	2778	0.2	228	0.04	9	18	25550	9-year AE

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Table C.7. POM Dermal Dose for MEIR

Dose-dermal[1]	Cs[2]	SA[3]	SL[4]	Ef[6]	ABS[6]	ED[7]	BW[8]	AT[9]	Exposure
5.24E-08	4.82	5500	1	350	0.13	70	63	25550	70-year HE[10]
3.10E-09	4.82	4700	0.2	121	0.13	70	63	25550	70-year AE[11]
2.25E-08	4.82	5500	1	350	0.13	30	63	25550	30-year HE
1.33E-09	4.82	4700	0.2	121	0.13	30	63	25550	30-year AE
1.31E-08	4.82	3044	1	350	0.13	9	18	25550	9-year HE
1.55E-09	4.82	2778	0.2	228	0.13	9	18	25550	9-year AE

Notes for Tables C.6 – C.7:

[1] Dose-dermal = dermal dose (mg/kg/day) in scientific notation (i.e. 1.0E-02 = 0.01)
[2] Cs = soil concentration (µg/kg)
[3] SA = surface area of exposed skin (cm^2)
[4] SL = soil loading on skin (mg/cm^2-day)
[5] ABS = fraction absorbed across the skin
[6] BW = body weight (kg)
[7] EF = exposure frequency (days/year)
[8] ED = exposure duration (years)
[9] AT = averaging time (e.g., 25550 days for 70 years for cancer risk)
[10] HE = high exposure
[11] AE = average exposure

Soil Ingestion Doses for Maximum Exposed Individual Resident (MEIR)

Tables C.8 and C.9 give the soil ingestion doses for POM, arsenic, nickel, chromium 6+, and formaldehyde at the MEIR location. Notes explaining the column heading abbreviations are included after Table C.9.

Table C.8. Arsenic Soil Ingestion Dose for MEIR

Dose-ing-soil[1]	Csoil[2]	GRAF[3]	SIR[4]	EF[5]	ED[6]	AT[7]	Exposure
7.77E-08	47.6915406	1	1.7	350	70	25550	70-year
3.33E-08	47.6915406	1	1.7	350	30	25550	30-year
5.12E-08	47.6915406	1	8.7	350	9	25550	9-year

Table C.9. POM Soil Ingestion Dose for MEIR

Dose-ing-soil[1]	Csoil[2]	GRAF[3]	SIR[4]	EF[5]	ED[6]	AT[7]	Exposure
7.86E-09	4.819	1	1.7	350	70	25550	70-year
3.37E-09	4.819	1	1.7	350	30	25550	30-year
5.17E-09	4.819	1	8.7	350	9	25550	9-year

Notes for Tables C.8 – C.9:

[1] Dose-ing-soil	= dose from soil ingestion (playground setting) (mg/kg body weight * day) in scientific notation
[2] Csoil	= concentration of contaminant in soil (µg/kg)
[3] GRAF	= gastrointestinal relative absorption fraction
[4] SIR	= soil ingestion rate (mg/kg body weight * day)
[5] EF	= exposure frequency (days/year)
[6] ED	= exposure duration (years)
[7] AT	= averaging time (e.g., 25550 days for 70 years for cancer risk)

Plant Ingestion Doses for Maximum Exposed Individual Resident (MEIR)

Tables C.10 and C.11 give the plant ingestion doses for POM, arsenic, nickel, chromium 6+, and formaldehyde at the MEIR location. Notes explaining the column heading abbreviations are included after Table C.11.

Table C.10. Arsenic Plant Ingestion Dose for MEIR

Dose-plants[1]	Cf[2]	IP[3]	GRAF[4]	L[5]	EF[6]	ED[7]	AT[8]	Plant Type	Exposure[9, 10]
9.44E-08	0.061931289	10.6	1	0.15	350	70	25550	leafy	70 yr HE
1.92E-09	0.001271774	10.5	1	0.15	350	70	25550	root	70 yr HE
2.01E-09	0.002861492	4.88	1	0.15	350	70	25550	protected	70 yr HE
4.83E-08	0.027741622	12.1	1	0.15	350	70	25550	exposed	70 yr HE
2.58E-08	0.061931289	2.9	1	0.15	350	70	25550	leafy	70 yr AE
5.78E-10	0.001271774	3.16	1	0.15	350	70	25550	root	70 yr AE
5.72E-10	0.002861492	1.39	1	0.15	350	70	25550	protected	70 yr AE
1.42E-08	0.027741622	3.56	1	0.15	350	70	25550	exposed	70 yr AE
4.05E-08	0.061931289	10.6	1	0.15	350	30	25550	leafy	30 yr HE
8.23E-10	0.001271774	10.5	1	0.15	350	30	25550	root	30 yr HE
8.60E-10	0.002861492	4.88	1	0.15	350	30	25550	protected	30 yr HE
2.07E-08	0.027741622	12.1	1	0.15	350	30	25550	exposed	30 yr HE
1.11E-08	0.061931289	2.9	1	0.15	350	30	25550	leafy	30 yr AE
2.48E-10	0.001271774	3.16	1	0.15	350	30	25550	root	30 yr AE
2.45E-10	0.002861492	1.39	1	0.15	350	30	25550	protected	30 yr AE
6.09E-09	0.027741622	3.56	1	0.15	350	30	25550	exposed	30 yr AE
1.25E-08	0.061931289	10.9	1	0.15	350	9	25550	leafy	9 yr HE
3.50E-10	0.001271774	14.9	1	0.15	350	9	25550	root	9 yr HE
3.52E-10	0.002861492	6.66	1	0.15	350	9	25550	protected	9 yr HE
8.05E-09	0.027741622	15.7	1	0.15	350	9	25550	exposed	9 yr AE
3.34E-09	0.061931289	2.92	1	0.15	350	9	25550	leafy	9 yr AE
9.60E-11	0.001271774	4.08	1	0.15	350	9	25550	root	9 yr AE
8.63E-11	0.002861492	1.63	1	0.15	350	9	25550	protected	9 yr AE
2.13E-09	0.027741622	4.16	1	0.15	350	9	25550	exposed	9 yr AE

Table C.11. POM Plant Ingestion Dose for MEIR

Dose-plants[1]	Cf[2]	IP[3]	GRAF[4]	L[5]	EF[6]	ED[7]	AT[8]	Plant Type	Exposure[9, 10]
1.23E-07	0.080885198	10.6	1	0.15	350	70	25550	leafy	70 yr HE

1.64E-09	0.001084068	10.5	1	0.15	350	70	25550	root	70 yr HE
7.61E-10	0.001084068	4.88	1	0.15	350	70	25550	protected	70 yr HE
7.21E-08	0.041427888	12.1	1	0.15	350	70	25550	exposed	70 yr HE
3.37E-08	0.080885198	2.9	1	0.15	350	70	25550	leafy	70 yr AE
4.93E-10	0.001084068	3.16	1	0.15	350	70	25550	root	70 yr AE
2.17E-10	0.001084068	1.39	1	0.15	350	70	25550	protected	70 yr AE
2.12E-08	0.041427888	3.56	1	0.15	350	70	25550	exposed	70 yr AE
5.29E-08	0.080885198	10.6	1	0.15	350	30	25550	leafy	30 yr HE
7.02E-10	0.001084068	10.5	1	0.15	350	30	25550	root	30 yr HE
3.26E-10	0.001084068	4.88	1	0.15	350	30	25550	protected	30 yr HE
3.09E-08	0.041427888	12.1	1	0.15	350	30	25550	exposed	30 yr HE
1.45E-08	0.080885198	2.9	1	0.15	350	30	25550	leafy	30 yr AE
2.11E-10	0.001084068	3.16	1	0.15	350	30	25550	root	30 yr AE
9.29E-11	0.001084068	1.39	1	0.15	350	30	25550	protected	30 yr AE
9.09E-09	0.041427888	3.56	1	0.15	350	30	25550	exposed	30 yr AE
1.63E-08	0.080885198	10.9	1	0.15	350	9	25550	leafy	9 yr HE
2.99E-10	0.001084068	14.9	1	0.15	350	9	25550	root	9 yr HE
1.33E-10	0.001084068	6.66	1	0.15	350	9	25550	protected	9 yr HE
1.20E-08	0.041427888	15.7	1	0.15	350	9	25550	exposed	9 yr AE
4.37E-09	0.080885198	2.92	1	0.15	350	9	25550	leafy	9 yr AE
8.18E-11	0.001084068	4.08	1	0.15	350	9	25550	root	9 yr AE
3.27E-11	0.001084068	1.63	1	0.15	350	9	25550	protected	9 yr AE
3.19E-09	0.041427888	4.16	1	0.15	350	9	25550	exposed	9 yr AE

Notes for Tables C.10 – C.11:

[1] Dose-plants = exposure dose through ingestion of plants (mg/kg/day) in scientific notation
[2] Cf = concentration in plant type (μg/kg)
[3] IP = consumption of plants (g/kg/day)
[4] GRAF = gastrointestinal relative absorption factor
[5] L = fraction of homegrown produce
[6] EF = exposure frequency (days/year)
[7] ED = exposure duration (years)
[8] AT = averaging time (e.g., 25550 days for 70 years for cancer risk)
[9] HE = high exposure
[10] AE = average exposure

Inhalation Cancer Risk for Maximum Exposed Individual Resident (MEIR)

Figure C.1 shows the inhalation cancer risk for POM, arsenic, nickel, chromium 6+, and formaldehyde at the MEIR location.

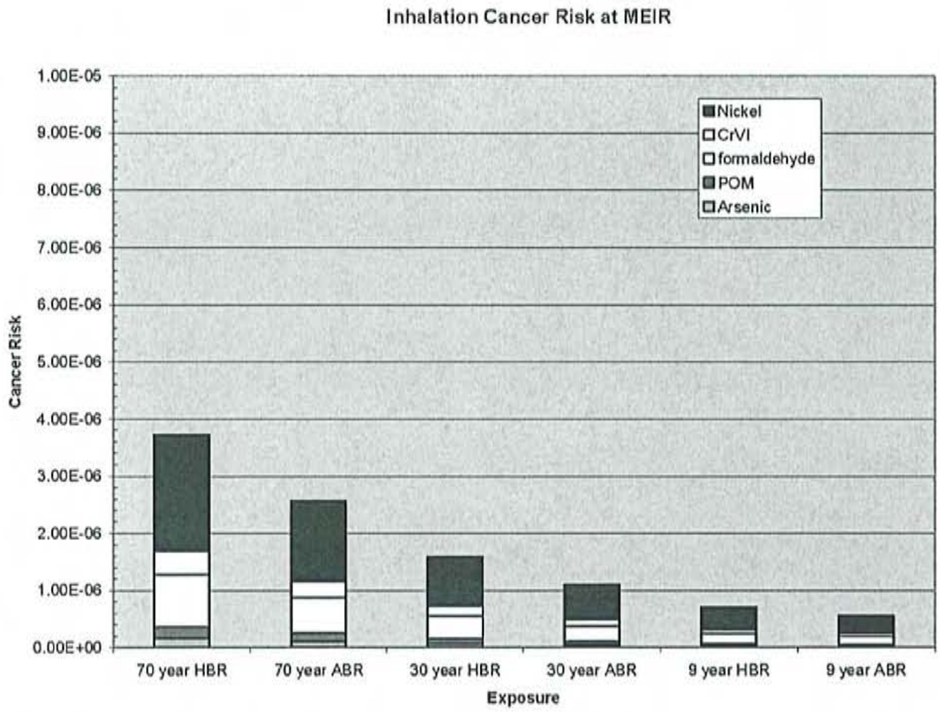

Figure C.1. Inhalation risk at MEIR location for contaminants of concern. (HBR = high breathing rate, and ABR = average breathing rate.)